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IRVINE

Taking Context to Heart: Momentary Emotions, Menstrual Cycle, and Cardiac Autonomic Regulation

THESIS

submitted in partial satisfaction of the requirements
for the degree of

MASTER OF ARTS

in Social Ecology

by

Shauna G. Simon

Thesis Committee:
Professor Larry D. Jamner, Chair
Assistant Professor Jenna Riis
Distinguished Professor Julian F. Thayer

2019

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ABSTRACT OF THE THESIS

Taking Context to Heart: Momentary Emotions, Menstrual Cycle, and Cardiac Autonomic Regulation

By

Shauna G. Simon

Master of Arts in Social Ecology

University of California, Irvine, 2019

Professor Larry D. Jamner, Chair

Background. Emotions have long been discussed in conjunction with the autonomic nervous system. Most research on heart-mood linkages does not consider sex differences or an evident underlying mechanism for sex differences- sex hormone levels. Further, most research is limited to cross-sectional and laboratory studies. The degree to which affect-autonomic associations manifest in everyday life may be different and may vary by sex and, for women, by menstrual cycle phase. This study employs ambulatory monitoring of cardiovascular measures (e.g., heart rate and heart rate variability; HRV) and concurrent emotions (e.g., sadness, stress, anxiety, anger, and happiness) in everyday life to better characterize affect-autonomic associations as a function of sex and menstrual cycle phase. Methods. Participants (N = 174) were monitored over a 5-day observation period (one 2-day and one 3-day session), using an ambulatory 24-hour electrocardiogram to monitor heart rate and ecological momentary assessment to record emotions every approximately 30 minutes. Women were monitored in both the early to mid-follicular and -luteal phases of their menstrual cycles and men in two comparably distanced sessions. Results. Multilevel models indicated that for both sexes, negative emotions and happiness were associated with elevated heart rate. Relative to men, women exhibited elevated

heart rate and reduced HRV during reports of anger. For women, during the luteal phase, momentary reports of sadness, stress, and anxiety were found to predict increased heart rate and reduced HRV. Conclusion. These findings demonstrate the importance of considering sex and menstrual cycle phase in research investigating heart-mood linkages.

Introduction

The association between emotions and autonomic responses have long been a source of investigation. The concept of a heart-mood link dates back over a century with Claude Bernard suggesting an interactive, bidirectional relation between cerebral arousal and autonomic control of the heart. Darwin, referencing Bernard's work, stated: "[…] when the heart is affected it reacts on the brain; and the state of the brain again reacts through the pneumo-gastric nerve on the heart; so that under any excitement there will be much mutual action and reaction between these" (Darwin, 1872). More recently, Thayer and colleagues proposed the Neurovisceral Integration Model (NIM), which at its core provides as a conceptual and theoretical framework integrating communication between the brain and the heart. The NIM explains that physiological measures such as HRV provide as an index for an organism's ability to continuously assess and respond to environmental threat or challenge. The NIM illustrates the integration of autonomic and affective systems for self-regulation and adaptation to environmental demands by suggesting neuroanatomical pathways between the ANS and brain regions responsible for emotion regulation, such as the amygdala and ventromedial prefrontal cortex (Julian F. Thayer, Åhs, Fredrikson, Sollers, & Wager, 2012; J. F. Thayer & Lane, 2000). In applying the conceptual framework of the NIM, research supports heart rate variability (HRV) as a proxy for emotional responding, such that higher overall HRV is associated with a greater stability of affective states and improved emotional outcomes (Sloan et al., 2017). While heart rate reflects levels of general nervous system arousal, HRV is thought to reflect the individual's capacity to transition from high to low arousal states. HRV is largely dependent on heart rate, as HRV should increase as the R-R interval increases, reflecting decreased heart rate (Monfredi et al., 2014). A recent meta-analysis of 24 studies concluded that HRV training resulted in large reductions in self-reported

stress and anxiety ratings (Goessl, Curtiss, & Hofmann, 2017). Conversely, reduced HRV has been associated with greater daily negative affect, increased attention to negative stimuli, depressive symptoms, and anxiety disorders (Conrad, Wilhelm, Roth, Spiegel, & Taylor, 2008; Gorman & Sloan, 2000; Kemp et al., 2010; Krypotos, Jahfari, van Ast, Kindt, & Forstmann, 2011; Shapiro, Jamner, Goldstein, & Delfino, 2001; Sin, Sloan, McKinley, & Almeida, 2016). Correspondingly, experiences of negative emotions (e.g., anger and anxiety) have been associated with increases in heart rate and blood pressure (Shapiro, Jamner, Goldstein, & Delfino, 2001).

These associations between affective and autonomic responding seem to show some degree of sex specificity. In general, men and women differ in hemodynamic responses to stress. Men demonstrate greater blood pressure responses, whereas women generally show greater changes in cardiac output and heart rate (Allen, Stoney, Owens, & Matthews, 1993). Thus, women do not demonstrate fight-or-flight patterns of activation in response to stress, rather they down-regulate the sympathetic network and hypothalamus-pituitary-adrenal (HPA) axis response to stress. This inhibition of sympathetic activity may foster an affiliative response to stress, which is associated with higher oxytocin and estrogen-enhanced oxytocin release (Taylor et al., 2000). Thus, women in the luteal phase of the menstrual cycle (characterized by lower levels of estrogen than progesterone) may not down-regulate the sympathetic response to stress to the same extent as women in the follicular phase (characterized by higher levels of estrogen than progesterone). To date, only a handful of published studies have been designed to delineate the biobehavioral pathways through which variability in estrogen/progesterone levels contribute to affect-autonomic associations. Among the more informative findings in the literature are studies demonstrating a shift towards greater sympathetic activation during the luteal phase, compared to

the follicular phase, of women's menstrual cycles. In these studies, the shift to more sympathetic nervous system dominance and greater parasympathetic withdrawal were attributed to the neuro-modulatory actions of progesterone, estradiol and their level ratios. Accordingly, changes in estradiol levels are capable of influencing these response patterns. Specifically, research on premenopausal women has found increased HRV during the follicular phase and decreased HRV during the luteal phase (McKinley et al., 2009; Pestana, Mostarda, Silva-Filho, Salvador, & de Carvalho, 2018; Sato & Miyake, 2004; Tenan, Brothers, Tweedell, Hackney, & Griffin, 2014; von Holzen, Capaldo, Wilhelm, & Stute, 2016). Correspondingly, in animal models, administration of estrogen has been associated with increases in parasympathetic tone (Dent, Du, & Kingwell, 2002).

The findings described above notwithstanding, studies investigating menstrual cycle differences in cardiovascular responses to stress have been inconsistent across studies. Some studies found a greater heart rate response to a laboratory stressor during the luteal relative to the follicular phase (Childs, Dlugos, & De Wit, 2010; M. K. Lustyk, Douglas, Shilling, & Woods, 2012; M. K. B. Lustyk, Olson, Gerrish, Holder, & Widman, 2010; Manhem, Jern, Pilhall, Shanks, & Jern, 1991), while other studies found no differences across menstrual cycle phase (Espin, Villada, Hidalgo, & Salvador, 2019; Gordon & Girdler, 2014; Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999; M. K. B. Lustyk et al., 2010; Pico-Alfonso et al., 2007; Villada et al., 2017). Similarly, studies investigating menstrual cycle differences in HRV responses to stress have also shown inconsistent results. Sato and Miyake (2004) found greater HRV reactivity in women in the luteal phase compared to the follicular following a mental stress task, whereas others found no differences in HRV reactivity across the menstrual cycle following a stress paradigm (Pico-Alfonso et al., 2007; Zanten, Carroll, & Ring, 2009). Of note, these

studies were based on laboratory settings that vary in the type of stress induction task used. Most research investigating heart-mood linkages are limited to cross-sectional and laboratory studies that apply an emotion induction procedure (e.g., video clips, recall tasks, mental imagery, etc.). Discrepancies between these two bodies of research are likely due in part to the heterogeneity of what constitutes the emotional stressor across protocols, variables used to reflect autonomic and affective responsivity; and the reduced face validity associated with emotional experiences elicited in a controlled laboratory environment. Other limitations that might account for discrepancies between these laboratory studies include low ecological validity and clinical predictability. The degree to which these associations manifest in everyday life may be different. That is, the degree to which a specific stimulus elicits arousal likely differs by individual. One potential method for improving upon the ability to detect heart-mood linkages is through implementing ambulatory monitoring in everyday settings. Ecological momentary assessments (EMA), or the repeated sampling of behaviors and experiences in real time, offers the ability to assess cardiac control of the heart across a range of experiences with greater relevancy to individuals. For instance, in a study assessing objective physiology and self-reported mood using ambulatory methodology as well as traditional retrospective self-reports in breast cancer patients, momentary levels of mood were related to concurrent heart rate and respiratory measures, whereas standard questionnaire responses reflected a retrospective bias (Grossman, Deuring, Garland, Campbell, & Carlson, 2008). The prognostic value of such monitoring is further demonstrated by ambulatory studies of blood pressure that have been shown to be even more predictive of morbidity and mortality than clinic-derived measures of blood pressure (Ohkubo et al., 2000). The present study utilizes ambulatory monitoring in a real-life setting in an attempt to better understand these inconsistencies in research on autonomic responding. Further, this study

extends these findings beyond stress reactivity to autonomic responding associated with several discrete emotional states (sadness, anxiety, stress, anger, and happiness). To the best of our knowledge, no study has investigated menstrual cycle phase effects on the association between heart rate and HRV to discrete emotional states in everyday life.

Aside from the conceptual and theoretical reasons for studies looking at mood-heart associations and the moderating effects of sex and menstrual cycle, this research is significant from a clinical perspective because of the role of emotion in vulnerability for cardiovascular disease (CVD). Increased HRV has been related to a healthier cardiac state, whereas reductions in HRV encompass parasympathetic withdrawal, an autonomic state that when consistent overtime, is correlated with increased cardiovascular risk (Schuster, Fischer, Thayer, Mauss, & Jarczok, 2016). Depression, anxiety, and hostility increase the risk of developing cardiovascular disease CVD two-to four times (Carney, Freedland, Miller, & Jaffe, 2002; Chida & Steptoe, 2009; Cuttler et al.; Kemp, Quintana, Felmingham, Matthews, & Jelinek, 2012; Sirois & Burg, 2003). Mood disorders are more prevalent in woman than men, and depression increases risk for CVD to a greater extent in women (Möller-Leimkühler, 2007). Research suggests that these associations are at least partially explained by a greater reactivity to psychosocial stressors related to CVD and depression (Moller-Leimkuhler, 2010; Wright, Simpson, LVan Lieshout, & Steiner, 2014). Before menopause, sex hormone levels seem to protect women from such stressors even though they are more physiologically reactive, as exposure to stress does not seem to accelerate CVD progression. Relative to men, women are delayed in the age of heart attacks, with men demonstrating a two-fold increase in risk of developing CVD compared to premenopausal women. Converging lines of evidence suggest that female protection against CVD is partly due to sex hormone levels. For instance, once sex hormone levels reduce during

menopause, women rapidly match the CVD rates of men and premenopausal women with premature ovarian failure or an oophorectomy have a comparable risk for CVD (Parker, Jacoby, Shoupe, & Rocca, 2009). Postmenopausal women using hormone replacement therapy show large reductions in coronary artery risk (Barrett-Connor, 1996). Even research using animal models and in vitro preparations have found that estrogen has vasoprotective effects (Xing, Nozell, Chen, Hage, & Oparil, 2009).

The present study employs ambulatory monitoring of autonomic regulation of the heart and reports of emotions to investigate the association between momentary changes in heart rate and HRV and contemporaneous emotions during everyday experiences across two phases of the menstrual cycle. Moreover, the current study takes into account sex differences in autonomic functioning, including sex as a moderator. Finally, this study applies female-only analyses to assess menstrual cycle as a mechanism for the proposed sex differences. Specifically, proposing that in women, the luteal phase could magnify the association between momentary changes in cardiac autonomic activity and emotions. This extends upon an earlier study that used a subset of the current study's cohort of women to assess measures of HRV during the luteal phase compared to the follicular phase (McKinley et al., 2009). Moreover, the results of the previous study did not report affective self-reports collected during the ambulatory monitoring periods, preventing an examination of affect-autonomic associations across the menstrual cycle. To our knowledge, no study to date has investigated differences in healthy adults across cardiac activity and momentary emotional states in daily life as a function of menstrual cycle phase, as this requires a longitudinal design that allows for stratification of women by menstrual cycle phase. Using this rigorous approach to capturing the association between cardiovascular measures, emotions, and menstrual cycle phase, we predicted that:

1. Measures of HRV and heart rate would be associated with reports of sadness, stress, anxiety, anger, and happiness. Specifically, during experiences of negative emotions and happiness, individuals would be more likely to exhibit increases in heart rate and reductions in HRV.
2. Relative to men, women would show stronger associations between autonomic indices and emotions. Specifically, women would be more likely to exhibit increases in heart rate and reductions in HRV when experiencing negative emotions.
3. Relative to the follicular phase, the luteal phase would be characterized by stronger affect-autonomic linkages. During the luteal phase, women would show larger increases in heart rate and reductions in HRV when experiencing negative emotions.

Methods

Participants

Participants were 174 healthy adults (87 female) ages 18 to 46 (31.23 ± 6.49) who were originally recruited to participate in a study of the cardiac autonomic consequences of negative interpersonal interactions. The ethnic breakdown was 58.6% White/Caucasian, 23.0% Asian, 11.5% Black/African American, 1.2% Hispanic, and 5.2% Other. Exclusion criteria included the following: body mass index of greater than 30 kg/m^2, known preexisting CVD or hypertension, taking any medication to regulate blood pressure, active psychiatric disease, diabetes, drug or alcohol abuse, or any other condition/regular use of medication likely to influence the ANS. In addition, women-specific exclusion criteria included the following: postmenopausal, pregnant, taking oral contraceptives, tubal ligations, history of menstrual cycle irregularities, menstrual cycle-related disorders (e.g., premenstrual syndrome, dysmenorrhea), or menstrual cycle length of less than 26 days or longer than 32 days. The study sample was composed of participants enrolled from southern California and New York metropolitan areas, recruited through flyers and

internet advertisements. The study was approved by the Institutional Review Boards of both sponsoring universities and all participants provided written informed consent.

Procedure

Participants underwent an orientation session during which demographic, health, and psychological characteristics were assessed. After signing informed consent, women began completing daily menstrual symptom ratings as the first phase of the study. Daily symptom ratings were used to confirm self-reported menstrual cycle length, estimate when to schedule female participants within each woman's cycle, and to exclude participants whose reports reflected menstrual cycle-related disorders. Participants were then monitored over a two-day followed by a three-day ambulatory monitoring session, where order of the sessions (two versus three days) was randomized. The start of all monitoring sessions were scheduled between 7:00AM and 11:00AM, during which participants were outfitted with an ambulatory 24-hour electrocardiogram (ECG) to monitor heart rate and a palm computer-based electronic diary (*e*Diary) to record emotions, interpersonal interactions, and activities. On each diary entry occasion, participants also rated the level of inconvenience associated with completing that entry on a 5-point scale ranging from 0 (not at all) to 4 (very much). The average level of diary inconvenience was found to be 1.27 (±0.95), corresponding to slightly more than "*Just a Little*" and indicated that participant burden was perceived as low and unlikely generated reactance.

Menstrual Cycle Monitoring

Following procedures used successfully in prior studies, the menstrual cycles of female participants were tracked using the Prospective Record of the Impact and Severity of Menstrual Symptoms calendar (PRISM; Reid, 1985). Responses on the PRISM determined the scheduling windows that defined the follicular and mid-luteal phases of participants' menstrual cycles as to

initiate ovulation testing for luteinizing hormone surges from urine samples (7-day Clearblue Easy Read Ovulation Test; Swiss Precision Diagnostics GmbH, Geneva Switzerland). Female participants were randomized to either undergo their first 2-3 day EMA monitoring session during the follicular phase (days 3 to 8, post-menstruation) and their second 2-3 day EMA monitoring session in the mid-luteal phase (5 to 10 days post-ovulation) of their menstrual cycles or in the reverse order. Male participants maintained a comparable symptom diary to the PRISM with comparable time intervals between EMA sessions. Order was included as a covariate in all models. The protocol for menstrual cycle phase scheduling is illustrated in Figure 1.

Cardiovascular Measures

To measure heart rate and HRV, participants' ECGs were continuously recorded at 200Hz using a three-lead configuration with a LifeShirt (VivoMetrics, Ventura, CA). Participants were to wear the LifeShirt throughout the 2-3 day recording session but were permitted to remove the device as needed (e.g., for bathing). If ECG recording was of poor quality in 20% or more of *e*Diary occasions in a day, then that day was excluded from analysis. ECG data were stored on the recorder and later uploaded to a secure server for storage and analysis. The digitized ECG waveforms were exported from Vivologic software to custom-written event detection software to produce an RR interval series. Errors in detection of R-waves were corrected by visual inspection and ectopic beats were corrected by interpolation. From the RR interval series, three outcome variables were computed: average heart rate (average beats per minute), root mean squared successive difference (RMSSD), and high frequency (HF power) RR interval variability. Average heart rate reflects increased arousal, reflecting stimulation of the sympathetic branch or parasympathetic withdrawal. Heart rate in the HF band consists of power spectral analysis of heart rate 0.15 to 0.5 hertz range. According to the Porges-Bohrer method for

quantifying vagal tone, the HF power reflects parasympathetic activation, or vagally-mediated HRV, as it conveys HRV related to the respiratory cycle (Laborde, Mosley, & Thayer, 2017; Lewis, Furman, McCool, & Porges, 2012). The vagal index of HF power was highly correlated with the time-domain measure of RMSSD ($r = .98$, $p < .001$).

Daily Diary Measures

The application of EMA allows for multiple assessments over time, thus decreasing the effect of retrospective biases by reducing the time frame of assessment (prompting participants approximately every 30 minutes). The *e*Diary consisted of a customized program installed on Palm Tungsten PDAs (palmOne, Milpitas, CA). In order to accommodate with participants' schedules, the *e*Diary was preprogrammed to accept up to four daily "time-out" periods, during which signaling could be suppressed. To promote adherence, project staff contacted participants on the evening of the first day of monitoring to review successes, issues, and questions regarding the diary protocol. Monitoring days with less than 75% *e*Diary compliance were excluded from analysis. Using the *e*Diary, participants reported ratings of stress, anxiety, sadness, anger, and happiness on a 4-point Likert scale[1]. Emotion ratings were coded as 0 for absence of emotion and 1 for experience of emotion. Although not included in this study, the diary collected information on interpersonal interactions, as social processes have been frequently associated with HRV (Kok & Fredrickson, 2010). Participants reported being alone in 64.50% of entries. Men reported greater alone time (67.05%) than women (61.95%), but time alone for women did not differ by menstrual cycle (follicular phase = 61.72%; luteal phase = 61.74%). Other queries

[1] For brevity, emotions are referred to as stress, anxiety, sadness, and anger. It should be noted that the original scales for anxiety, sadness, and anger consisted of the phrasing "nervousness/worry", "sadness/disappointment", and "anger/frustration".

included in the diary consisted of information on location, activities, and consumption of cigarettes, alcohol, and caffeine.

Data Reduction and Analysis Plan

Multilevel models were used to investigate the associations between emotions and heart rate/HRV as well as the extent to which these potential associations vary by sex and menstrual cycle phase. Multilevel modeling accommodates the nested nature of the diary data in which diary entries are nested within participants. That is, multilevel models account for repeated measures and allow for within-subject dependence, varying observation points between participants, and separate yet simultaneous tests of within-person and between-persons associations. Models included random intercepts and random slopes to allow for within-person emotion levels to differ between participants. Separate analyses were conducted for each discrete emotion (e.g., stress, sadness, anxiety, anger, or happiness) across each outcome (e.g., heart rate, RMSSD, or HF power). Distributions of HF power and RMSSD were positively skewed, as was determined by visual inspection and descriptive statistics. Thus, a natural logarithmic (ln) transformation was computed to correct for skewness. Relevant person-level variables (mean-centered age) and observation-level variables (posture and condition/order) were also included as covariates in all analyses to account for potential confounding, as previous literature on cardiovascular measures have demonstrated associations between these variables[2] (Murata, Landrigan, & Araki, 1992; Wahabi, Pouryayevali, Hari, & Hatzinakos, 2014; Yeragani, Sobolewski, Kay, Jampala, & Igel, 1997). Several model approaches and covariance matrices were compared, and improved fit was determined using Likelihood Ratios, Wald's tests, Akaike

[2] We evaluated the extent to which each covariate accounted for variance in heart rate, RMSSD, and HF Power. Sitting, standing, and walking predicted higher heart rate and lower HF Power and RMSSD relative to reclining (*p*s < .001). Age was also associated with RMSSD and HF Power, as increased age predicted lower RMSSD and HF Power (*p*s < .001).

information criterion (AIC) scores, and Bayesian information criterion (BIC) scores. All models assumed a Toeplitz variance-covariance matrix structure with two bands, implying temporal carryover within two adjacent diary entries. Maximum likelihood (ML) estimation was used in the model building in order to allow for testing of likelihood ratios needed to distinguish between nested models with varying fixed effects. Restricted maximum likelihood (REML) estimation was used in the final models reported below and implemented using STATA MIXED. For clarity, specific analytic procedures are detailed with each research question's results below, where all outcome variables will often be referred to as autonomic nervous system (ANS) outcomes. All analyses were conducted using Stata version 15 (StataCorp, 2017).

Results

Descriptive statistics

Participants responded to 17,661 total diary prompts, representing an average 91.25% completion rate. This is superior to the ~80% average completion rate for EMA studies (Csikszentmihalyi & Larson, 2014). The mean number of diary entries completed per day was 21.43 (SD = 4.43) and the mean number of total diary entries completed per participant was 101.50 (SD = 23.14). Adherence did not vary as a function of sex, menstrual cycle phase (for women), or order of experimental session. The mean number of study days was 4.74 (SD = 0.69), with 84% of participants completing five days, 9.1% four days, 4% three days, 2.3% two days, and 0.6% completing one day.

Participant demographics, characteristics, and intraclass correlations (ICC) for each outcome are provided in Table 2. Demographics related to age, income, BMI, and physiological indices are provided by sex. Men and women differed significantly in regard to average heart rate (p = .037), but not average RMSSD (p = .62), or average HF power (p = .53) .The ICC was

examined using null models and indicated that there is a significant amount of between person variance across heart rate, RMSSD, and HF power. ICC values for the three null models ranged from .34 to .42, which reflects the ratio between the variance due participants over the total variance. This indicates that multilevel analyses were warranted, as 34% - 42% of the variation in ANS outcomes was across participants, and the remaining 64%-58% [(1-ICC) * 100] was within participants.

Hypothesis 1. Measures of heart rate and HRV are associated with reports of emotions. During experiences of negative emotions and happiness, individuals would be more likely to exhibit increases in heart rate reductions in HRV.

Analyses. To address hypothesis 1, we assessed whether emotion (e.g., sadness, stress, anxiety, anger, or happiness) was associated with within-person measures of HRV and heart rate. The use of EMA to investigate the association between affect and ANS outcomes is valuable in that each person provides as their own control. Thus, our model assesses the deviations in negative affect which are associated with changes in ANS outcomes within a person.

$$\text{Level 1:} \qquad \text{ANS}_{ij} = \beta_{0j} + \beta_{1j}(\text{Emotion}_{ij}) + \beta_{2j}(\text{Posture}_{ij}) + e_{ij}$$

At level 1 (within-person level), the dependent variable (ANS_{ij}) represents momentary ANS outcome (e.g., heart rate, RMSSD, or HF power) of subject *j* at time *i*. This specifies a linear function of time. ANS outcome is modeled as a function of person-specific intercepts, (β_{0j}), associations with concurrent emotion (e.g., absence of sadness) at observation time *i* for participant *j* ($Emotion_{ij}$), associations with posture (e.g., walking, standing, sitting, or reclining) at time *i* for participant *j* ($Posture_{ij}$), and residual error (e_{ij}).

Level 2: $\beta_{0i} = \gamma_{00} + \gamma_{01}(\text{Age}_i) + \gamma_{02}(\text{Order}_i) + u_{0j}$
$\beta_{1i} = \gamma_{10} + \gamma_{11}(\text{Age}_i) + \gamma_{12}(\text{Order}_i) + u_{1j}$

At level 2 (between-person level), the intercept for participant *i* is determined by: (1) a fixed intercept for the population γ_{00}, (2) a fixed effect of person-level coefficients (Age_i and Order_i) for the population (γ_{01}, γ_{02}), and (3) a level-2 random effect or intercept deviation (u_{0j}). Similarly, the slope for participant *i* is determined by: (1) a fixed slope effect for the population (γ_{01}), (2) a fixed common effect of age and order on slopes γ_{12} and γ_{13}, and (3) a level-2 random effect or slope deviation (u_{1j}).

Results. Of all diary entries, participants reported feeling sadness in 14.14% of entries, stress in 36.56%, anxiety in 20.39%, anger in 15.20%, and happiness in 55.22% of entries. Findings related to the first hypothesis are presented in Table 3. As predicted, all emotions were associated with changes in heart rate. Such that, when participants reported high levels of sadness, stress, anxiety, anger or happiness, they experienced significant increases in heart rate (b_{sad} = .92, SE = .26, p < .001, 95%CI [.41, 1.43]; b_{stress} = .93, SE = .19, p < .001, 95%CI [.56, 1.30]; $b_{anxiety}$ = .77, SE = .22, p = .001, 95%CI [.33, 1.20]; b_{anger} = .82, SE = .22, p < .001, 95%CI [.38, 1.26]; b_{happy} = .69, SE = .19, p < .001, 95%CI [.32, 1.07]).

For models including stress, anger, and happiness, results demonstrated a significant reduction in HF power (b_{stress} = -.02, SE = .01, p = .024, 95%CI [-.03, <-.01]; b_{anger} = -.02, SE = .01, p = .026, 95%CI [-.04, <-.01]; b_{happy} = -.03, SE = .01, p < .001, 95%CI [-.04, -.01]) and a significant reduction in RMSSD (b_{stress} = -.02, SE = .01, p = .006, 95%CI [-.04, -.01]; b_{anger} = -.02, SE = .01, p = .033, 95%CI [-.04, <-.01]; b_{happy} = -.03, SE = .01, p = .001, 95%CI [-.04, -.01]). Overall, the results provided partial support of the hypothesis, as evidenced by significant effect of all emotions on heart rate and a significant effect of stress, anger, and happiness on

HRV. However, this did not extend to all emotions, as sadness and anxiety were not associated with changes in RMSSD or HF power.

Hypothesis 2. Relative to men, women are more likely to experience increases in heart rate and decreases in HRV when experiencing negative emotions

Analyses. Analyses for the second hypothesis assessed whether sex moderates the association between momentary emotions and ANS outcomes. Separate analyses were again conducted for each emotion and its association with each ANS outcome. At level 1, repeated measures of ANS outcome (ANS_{ij}) is modeled as a function of: person-specific intercepts (β_{0j}), residual error (e_{ij}), and associations with emotion ($Emotion_{ij}$), the interaction between emotion and sex (Emotion * Sex_{ij}), and posture ($Posture_{ij}$). At level 2, the intercept and slope for participant *i* are determined as they were in hypothesis 1, with the addition of sex (0 = men, 1 = women) as an additional fixed factor.

Level 1: $$\mathrm{ANS_{ij}} = \beta_{0j} + \beta_{1j}(\mathrm{Emotion_{ij}}) + \beta_{2j}(\mathrm{Emotion} * \mathrm{Sex_{ij}}) + \beta_{3j}(\mathrm{Posture_{ij}}) + e_{ij}$$

Level 2: $$\beta_{0i} = \gamma_{00} + \gamma_{01}(\mathrm{Age_i}) + \gamma_{02}(\mathrm{Order_i}) + \gamma_{04}(\mathrm{Sex_i}) + u_{0j}$$
$$\beta_{1i} = \gamma_{10} + \gamma_{11}(\mathrm{Age_i}) + \gamma_{12}(\mathrm{Order_i}) + \gamma_{14}(\mathrm{Sex_i}) + u_{1j}$$

Results. Of the five emotion states tested in the second hypothesis, there was a significant sex by emotion interaction for models including anger and happiness (see Table 4). Sex significantly moderated the effect of anger and happiness on heart rate (b_{anger} = 1.09, SE = .45, p = .015, 95%CI [.21, 1.96]); b_{happy} = -1.17, SE = .38, p = .002, 95%CI [-1.91, -.43]), on RMSSD (b_{anger} = 1.09, SE = .45, p = .015, 95%CI [.21, 1.96]); b_{happy} = -1.17, SE = .38, p = .002, 95%CI [-1.91, -.43]), and on HF power (b_{anger} = 1.09, SE = .45, p = .015, 95%CI [.21, 1.96]); b_{happy} = -1.17, SE = .38, p = .002, 95%CI [-1.91, -.43]). The simple slopes for models predicting

heart rate, illustrated in Figure 2, revealed that women exhibited greater increases in heart rate during episodes of anger relative to men. Correspondingly, as depicted in Figures 3 and 4, women exhibited significantly decreased RMSSD and HF power during experiences of anger, whereas the simple slopes for men reveal slight increases in RMSSD and HF power, although not significant. On the contrary, during experiences of happiness, men exhibited increased heart rate, whereas women showed no differences in heart rate during reports of happiness (see Figure 5). Men also demonstrated increased RMSSD and HF power during episodes of happiness, whereas women did not exhibit changes in HRV during reports happiness (see Figures 6 and 7).

Hypothesis 3. During the luteal phase, women are more likely to exhibit stronger associations between emotion and autonomic indices. Specifically, women are more likely to show increases in heart rate and reductions in HRV when experiencing negative emotions relative to the follicular phase.

Analyses. Analyses to assess whether menstrual cycle phase moderates the association between emotion and ANS outcomes were used to address hypothesis 3. Due to evident sex differences, all analyses for hypothesis 3 were estimated solely for women. Separate analyses were again conducted for each emotion and its association with each ANS outcome. At level 1, repeated measures of ANS outcome (ANS_{ij}) is modeled as a function of: person-specific intercepts (β_{0j}), residual error (e_{ij}), and associations with emotion ($Emotion_{ij}$), menstrual cycle phase ($Phase_{ij}$), the interaction between emotion and menstrual cycle phase (Emotion * $Phase_{ij}$), and posture ($Posture_{ij}$). At level 2, the intercept and slope for participant *i* are determined as they were in hypothesis 1.

Level 1: $\mathrm{ANS_{ij}} = \beta_{0j} + \beta_{1j}(\mathrm{Phase_{ij}}) + \beta_{2j}(\mathrm{Phase_{ij}}) + \beta_{3j}(\mathrm{Emotion} * \mathrm{Phase_{ij}}) + \beta_{4j}(\mathrm{Posture_{ij}}) + e_{ij}$

Level 2: $\beta_{0i} = \gamma_{00} + \gamma_{01}(\mathrm{Age_i}) + \gamma_{02}(\mathrm{Order_i}) + u_{0j}$
$\beta_{1i} = \gamma_{10} + \gamma_{11}(\mathrm{Age_i}) + \gamma_{12}(\mathrm{Order_i}) + u_{1j}$

Results. The mean number of diary entries completed per day for women did not differ by phase (p = .48). During the follicular phase, women completed an average of 21.56 (SD = 4.44) entries per day compared to an average of 21.30 (SD = 5. 18) during the luteal phase. Descriptive statistics for each emotion and cardiovascular variable by phase are provided in Table 5. Table 6 shows several significant interactions between emotion and menstrual cycle phase, demonstrating that emotions across follicular and luteal phases of the menstrual cycle were associated with changes in heart rate and RMSSD, and HF power. As hypothesized, these findings demonstrate that the effect of negative emotions on indices of cardiac autonomic activity were magnified during the luteal phase. Simple slopes, illustrated in Figures 8 – 10, revealed that relative to the follicular phase, women in the luteal phase exhibited significant increases in heart rate during experiences of sadness, stress, and anxiety (b_{sad} = 1.45, SE = .69, p = .037, 95%CI [.09, 2.81]; b_{stress} = 1.09, SE = .48, p = .022, 95%CI [.15, 2.02]; $b_{anxiety}$ = 1.47, SE = .56, p = .009, 95%CI [.37, 2.57]). Correspondingly, women in the luteal phase exhibited reductions in HRV during experiences of sadness, stress, and anxiety. Simple slopes, as depicted in Figures 11 – 13, revealed that women in the luteal phase had significant reductions in RMSSD when sad, stress, or anxious relative to the follicular phase (b_{sad} = -.10, SE = .03, p = .001, 95%CI [-.15, -.03]; b_{stress} = -.04, SE = .02, p = .048, 95%CI [-.15, -.04]; $b_{anxiety}$ = -.08, SE = .02, p = .001, 95%CI [-.01, .06]). Similarly, as illustrated in Figures 13 – 15, HF power significantly reduced during experiences of sadness, stress, or anxiety (b_{sad} = -.08, SE = .03, p = .006, 95%CI

[-.14, -.02]; b_{stress} = -.06, SE = .02, p = .004, 95%CI [-.09, -.02]; $b_{anxiety}$ = -.08, SE = .02, p = .001, 95%CI [< -.01, .06]).

Thus, models including sadness, stress, and anxiety demonstrated support for the hypothesis that women in the luteal phase of the menstrual cycle would show stronger associations during experiences of negative emotions. Specifically, relative to the follicular phase, the luteal phase was characterized by increases in heart rate and decreases in HRV during reports of sadness, stress, or anxiety. Models including stress and anger did not support the original hypothesis, as this was likely due to robust sex differences demonstrated in hypothesis 2 above. Importantly, for models including stress, sadness, and anxiety, the main effects of cycle phase and emotion on heart rate or HRV became non-significant after including the interaction between concurrent emotion and luteal phase. This suggests that the experience of both negative emotions (stress, sadness, and anxiety) and increased progesterone levels (during the luteal phase) uniquely predict reductions in RMSSD or HF power, beyond the effects of negative emotion or luteal phase alone. findings for models including anger or happiness were not sufficient to support the hypothesis.

Hypothesis 4. During the luteal phase, women are more likely to exhibit negative emotions.

Exploratory analyses of menstrual cycle phase and emotions were conducted in order to examine whether the associations between luteal phase, emotions, and cardiac autonomic indices were influenced by prevalence of premenstrual symptoms such as greater likelihood to experience negative emotions. Logistic multilevel models testing the association between menstrual cycle phase and emotions revealed that relative to the follicular phase, the odds of women reporting sadness, stress, and anxiety were significantly lower during the luteal phase (Figure 16). That is, during the follicular phase, women were 1.72 times more likely to

experience sadness (OR = 1.72, SE = .13, $p < .001$, 95% CI [1.49, 1.98]), 1.45 times more likely to experience stress (OR = 1.45, SE = .08, $p < .001$, 95% CI [1.31, 1.61]), and 1.44 times more likely to experience anxiety (OR = 1.44, SE = .09, $p < .001$, 95% CI [1.28, 1.63]). Although these findings run counter to the widely held belief of greater emotionality in the time leading up to menses, these results reveal the unique contribution of menstrual cycle phase and negative emotions on cardiovascular indices. That is, although women in the luteal phase were less likely to report sadness, stress, and anxiety, they were more physiologically sensitive to experiences of these emotions.

Discussion

This current study sought to determine (1) whether experiences of sadness, stress, anxiety, anger and happiness were associated with reductions in HRV and increases in heart rate, (2) whether this effect would be greater in women relative to men, and (3) among women, whether this effect would be greater in the luteal phase of the menstrual cycle.

Findings demonstrated that collapsing across sex, when individuals experienced sadness, stress, anxiety, anger, and happiness, they exhibited increases in heart rate. These findings are consistent with previous studies investigating negative affect and heart rate throughout the day (Carels, Blumenthal, & Sherwood, 2000; Sin et al., 2016; Sloan et al., 1994). This suggests that collapsing across sex, all discrete emotions elicit some level of physiological arousal. In contrast to emotion-heart rate associations, associations between HRV and emotions were more differentiated. Stress, anger and happiness were associated with reductions HRV, whereas sadness and anxiety were not linked to changes in HRV. These findings elucidate the importance of the arousal dimension of emotion within physiological responses. Consistent with previous research, anger, stress and happiness reflect high levels of arousal, whereas sadness consists of

lower levels of arousal (Russell & Mehrabian, 1977). Thus, although sadness and anxiety increase heart rate, they may not constitute a large enough threat to require halting the mobilization of resources (i.e., state of parasympathetic dominance). Perhaps during experiences of sadness, changes in heart rate are reflected more than changes in HRV because this emotion involves sympathetic activation in the absence of any greater parasympathetic withdrawal. Of note, anxiety has been proposed to include two subtypes: anxious apprehension and anxious arousal (Nitschke, 1998). While anxious apprehension reflects a state of worry, anxious arousal reflects a state of panic. Perhaps these results demonstrate greater anxious apprehension than anxious arousal, thus requiring less mobilization of resources than a state of anxiety related to panic.

Results from the statistical models addressing hypothesis 2 revealed that the effects of anger and happiness on heart rate and HRV were qualified by a significant interaction effect of emotion and sex. Relative to men, women exhibited reduced HRV and increased heart rate during experiences of anger and happiness. For anger, these results could be a manifestation of Western norms, as women more commonly apply emotional suppression (i.e., consciously decrease the behavioral expression of emotion) during episodes of anger, whereas men more commonly externalize anger. Suppression has been shown to increase sympathetic activation (e.g., skin conductance rate, finger pulse amplitude) (Gross, 1998a). More research is needed to better address sex differences in the mood-physiology link, particularly in regard to anger. Future work should consider suppression as a potential mediating factor. The lack of significant findings for an interaction effect of sex by sadness, stress, and anxiety is likely due to menstrual cycle effects. Although the interaction effects for menstrual cycle by emotion only include women, these findings might qualify the lack of significant sex by emotion findings for sadness, stress,

and anxiety because this study includes data from women in two extremes (follicular and luteal phase). We might expect to find sex differences if women were sampled randomly during a range of time periods throughout different phases of the menstrual cycle. On the other hand, to the extent to which autonomic mood associations were impacted by menstrual cycle phase, this design could also elucidate sex differences by including data from women in two extremes of their cycles. Future studies should investigate the extent to which consideration menstrual cycle phase exaggerates or impedes sex differences.

Results from models addressing hypothesis 3 partially support the first portion of the original hypothesis. Findings demonstrated that during the luteal phase, women exhibited increases in heart rate and decreases in HRV when sad, stressed, or anxious, but not during periods of anger and happiness. As expected, results for models predicting RMSSD and HF power showed similar results, as both are considered measures of HRV. Although both were included, RMSSD may be more germane to the process of HRV when measured in ambulatory conditions. Research has shown that RMSSD is not affected by changes in breathing patterns, whereas HF power decreases when changing from controlled to spontaneous breathing rates (Penttilä et al., 2001). Future research should consider the advantages of RMSSD over HF power in ambulatory conditions as a function of gender and sex.

Results from hypothesis 4 indicated that during the luteal phase, women do not experience greater emotionality. Interestingly, during the luteal phase, women were less likely to report sadness, stress, and anxiety. These findings seem counterintuitive and differ from a study employing similar EMA methodology that demonstrated no general effects of phase on mood effects (Davydov, Shapiro, & Goldstein, 2004). However, it is important to note that the current state of the literature on menstrual cycle/mood linkages is ambiguous. A recent meta-analysis of

prospective data studies reported that 8.5% of publications found evidence of women experiencing greater negative moods in a non-premenstrual phase (Romans, Clarkson, Einstein, Petrovic, & Stewart, 2012). Most importantly, the lack of increased emotionality during the luteal phase elucidates the unique effect of both negative emotions and luteal phase on heart rate and HRV.

Taken together, results from hypotheses 3 and 4 indicate that elevated estradiol levels (during the follicular phase) function as a cardioprotective mechanism, modulating physiological reactivity of negative emotions. Correspondingly, these results indicate that although women in the luteal phase might not exhibit greater instances of negative emotions, they could have a less flexible autonomic response to situational demands. This is in line with the recent focus on HRV as a proxy for emotion regulation, or the process of monitoring, evaluating, and modifying how an emotion is experienced or expressed (Gross, 1998b). Autonomic flexibility is considered to enhance emotion regulation efforts by allowing a greater capacity to modify physiological and emotional responses to changing situational demands (Appelhans & Luecken, 2006). As described earlier, HRV acts as a proxy for autonomic flexibility by reflecting the interplay between sympathetic and parasympathetic activity. According to the neurovisceral integration model, HRV reflects prefrontal control on subcortical activity, which allows for effective self-regulatory processes. Applying this to the current findings that demonstrate reduced HRV during negative emotional experiences in the luteal phase, women in the luteal phase could require more effort to engage in self-regulation of emotions.

This study has several limitations that should be acknowledged. The data reported in this study are from secondary analyses using data collected to answer different hypotheses than those included in this article. Therefore, the protocol was not designed to answer the current research

questions. For instance, menstrual cycle phase was not determined using serum hormone levels. However, the application of both daily PRISM reports and LH surge testing lend confidence to accurate follicular and luteal phase timing. In addition to measuring serum hormone levels for menstrual phase timing, future studies should investigate physiology as a function of momentary emotion and naturally-occurring estradiol and progesterone levels. Moreover, although the current study presents significant considerations for emotion-heart linkages, it is important to note that physiology is not synonymous with health. Future studies should apply a longitudinal design to identify whether the vulnerability to increased heart rate and decreased HRV during momentary negative emotions and luteal phase are predictive of greater risk of comorbidity of CVD and mood disorders following menopause.

In light of these limitations, these findings present methodological considerations for research as well as clinical implications. First, this study suggests that women in the luteal phase of the menstrual cycle could present a methodological issue for research on physiology, as these findings demonstrate that menstrual symptoms (e.g., changes in mood) could have effects on autonomic regulation of the heart. Second, there is growing evidence demonstrating associations between the ANS and a wide range of health conditions, including cardiovascular disease and increased morbidity and mortality (Julian F. Thayer, Yamamoto, & Brosschot, 2010). Thus, the current study suggests that women in the luteal phase could be more vulnerable to affect-mediated cardiovascular risk. This can inform future intervention efforts to mitigate low vagal tone during the luteal phase (e.g., breathwork practices).

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Tables

Table 1. *Percentage of diaries with emotion endorsed by sex and menstrual cycle*

	Sex		Menstrual Cycle Phase	
	Women	**Men**	**Follicular**	**Luteal**
Sadness	14.07%	14.21%	16.26%	11.49%
Stress	38.26%	34.85%	40.81%	35.88%
Anxiety	21.02%	19.76%	22.62%	18.85%
Anger	14.56%	15.84%	14.18%	15.25%
Happiness	55.39%	55.06%	54.80%	55.39%

Table 2. *Participant Demographics and Summary of Physiological Indices*

	Women (N = 87)		Men (N = 87)		All Participants
	M (SD)	**Min – Max**	***M(SD)***	**Min – Max**	**ICC**
Age	31.23 (6.49)	19 – 46	28.38 (6.98)	18 – 45	
BMI	24.43 (3.25)	16.30 – 36.21	24.43 (3.15)	19.37 – 35.53	
Income	59,481 (45,307)	0 – 250,000	49,934 (35,108)	0 – 150,000	
Physiological Variables					
HF Power	2.51 (.30)	1.76 – 3.23	2.51(0.36)	1.44 – 3.39	.37
RMSSD	3.39 (0.34)	2.47 – 4.29	3.41 (0.42)	2.17 – 4.42	.42
Heart rate	83.10 (7.24)	67.70 – 97.27	80.85 (9.19)	62.67 – 110.51	.34
Ethnicity		%			
White/Caucasian		58.6%			
Hispanic		1.2%			
Asian		23.0%			
Black/African American		11.5%			
Other		5.2%			
Unknown		.6%			

Table 3. *Heart rate and HRV associated with reported sadness, stress, anxiety, anger, and happiness*

	Heart Rate (bpm)			RMSSD			HF Power		
	b	SE*b*	*p*	*b*	SE*b*	*p*	*b*	SE*b*	*p*
Sadness	**.92**	**.26**	**< .001**	-.01	.01	.53	< -.01	.01	.75
Stress	**.93**	**.19**	**< .001**	**-.02**	**.01**	**.006**	**-.02**	**.01**	**.024**
Anxiety	**.77**	**.22**	**.001**	-.01	.01	.14	-.01	.01	.30
Anger	**.82**	**.22**	**< .001**	**-.02**	**.01**	**.033**	**-.02**	**.01**	**.026**
Happiness	**.69**	**.19**	**< .001**	**-.03**	**.01**	**.001**	**-.03**	**.01**	**.001**

Table 4. *Heart rate and HRV associated with the interaction between sex (1 = women, 0 = men) and momentary emotions*

	Heart Rate (bpm)			RMSSD			HF Power		
	b	SE*b*	*p*	*b*	S*Eb*	*p*	*b*	SE*b*	*p*
Negative Emotions									
Sadness*Women	-.04	.52	.94	-.01	.02	.73	< .01	.02	.98
Sadness	**.94**	**.38**	**.014**	< -.01	.02	.87	< -.01	.02	.81
Women	2.14	1.29	.10	.04	.06	.48	.05	.05	.27
Stress*Women	-.04	.38	.91	< -.01	.02	.92	< -.01	.02	.87
Stress	**.95**	**.28**	**.001**	-.02	.01	.07	-.03	.01	.16
Women	2.12	1.30	.10	.04	.06	.48	.05	.05	.26
Anxiety*Women	-.12	.44	.79	.01	.02	.64	.01	.02	.67
Anxiety	**.83**	**.33**	**.012**	-.02	.01	.18	-.01	.01	.31
Women	2.15	1.29	.10	.04	.06	.51	.06	.05	.28
Anger*Women	**1.09**	**.45**	**.015**	**-.06**	**.02**	**.001**	**-.05**	**.02**	**.007**
Anger	.25	.32	.44	.01	.01	.38	.01	.01	.68
Women	1.99	1.29	.12	.05	.01	.40	.06	.05	.21
Positive Emotions									
Happiness*Women	**-1.17**	**.38**	**.002**	**.04**	**.02**	**.004**	**.05**	**.02**	**.002**
Happiness	**1.28**	**.27**	**< .001**	**-.05**	**.01**	**< .001**	**-.05**	**.01**	**< .001**
Women	**2.81**	**1.31**	**.032**	.01	.06	.81	.03	.05	.60

Table 5. *Summary of physiological and emotion variables by menstrual cycle phase*

	Follicular Phase			Luteal Phase		
	***M* / %**	**SD**	**Min - Max**	***M* / %**	**SD**	**Min - Max**
Physiological Variables						
Mean heart rate (bpm)	82.80	8.31	65.48 – 108.01	83.37	7.30	65.92 – 103.05
Mean RMSSD	3.39	.39	2.25 – 4.47	3.40	.32	2.78 – 4.21
Mean HF Power (log)	2.51	.34	1.48 – 3.32	2.51	.28	1.98 – 3.26
Emotion Variables						
Sadness	16.26%			11.49%		
Stress	40.81%			35.88%		
Anxiety	22.62%			18.85%		
Anger	14.18%			15.25%		
Happiness	54.80%			55.39%		

Table 6. *Heart rate and HRV associated with the interaction between menstrual cycle phase (0 = follicular, 1 = luteal) and momentary emotions*

	Heart Rate (bpm)			RMSSD			HF Power		
	b	SE*b*	*p*	*b*	SE*b*	*p*	*b*	SE*b*	*p*
Negative Emotions									
Sadness*Luteal	**1.45**	**.69**	**.037**	**-.10**	**.03**	**.001**	**-.08**	**.03**	**.006**
Sadness	.35	.55	.44	.03	.02	.11	.03	.02	.11
Luteal	.34	.33	.29	.02	.01	.16	.01	.01	.45
Stress*Luteal	**1.09**	**.48**	**.022**	**-.04**	**.02**	**.048**	**-.06**	**.02**	**.004**
Stress	**.40**	**.34**	**.24**	< -.01	.01	.81	.01	.01	.54
Luteal	.13	.36	.72	.02	.01	.24	.02	.01	.16
Anxiety*Luteal	**1.47**	**.56**	**.009**	**-.08**	**.02**	**.001**	**-.08**	**.02**	**.001**
Anxiety	.08	.38	.83	.02	.02	.12	.03	.02	.07
Luteal	.21	.33	.53	.02	.01	.15	.02	.01	.22
Anger*Luteal	.72	.60	.23	-.05	.02	.06	-.03	.03	.26
Anger	**1.00**	**.41**	**.016**	-.03	.02	.14	-.03	.02	.09
Luteal	.36	.32	.26	.01	.01	.39	< .01	.01	.75
Positive Emotions									
Happiness*Luteal	-.14	.48	.77	.01	.02	.71	.03	.02	.06
Happiness	.18	.35	.60	-.01	.01	.63	**-.03**	**.01**	**< .001**
Luteal	.55	.41	.18	< .01	.02	.99	-.02	.01	.24

Figures

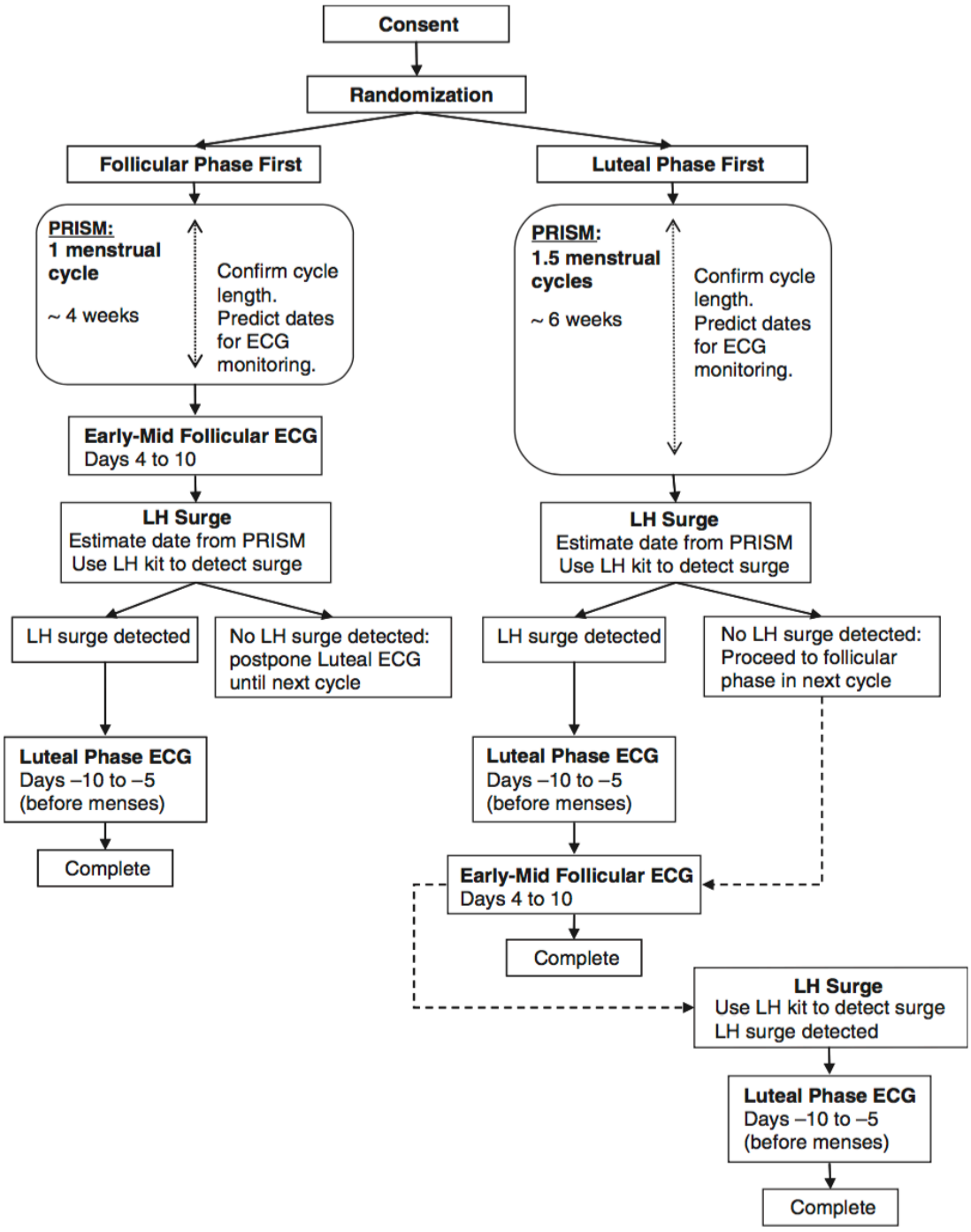

Figure 1. Study protocol: Menstrual phase tracking and timing of ECG monitoring. *PRISM* Prospective Record of the Impact and Severity of Menstrual Symptoms, *LH* luteinizing hormone

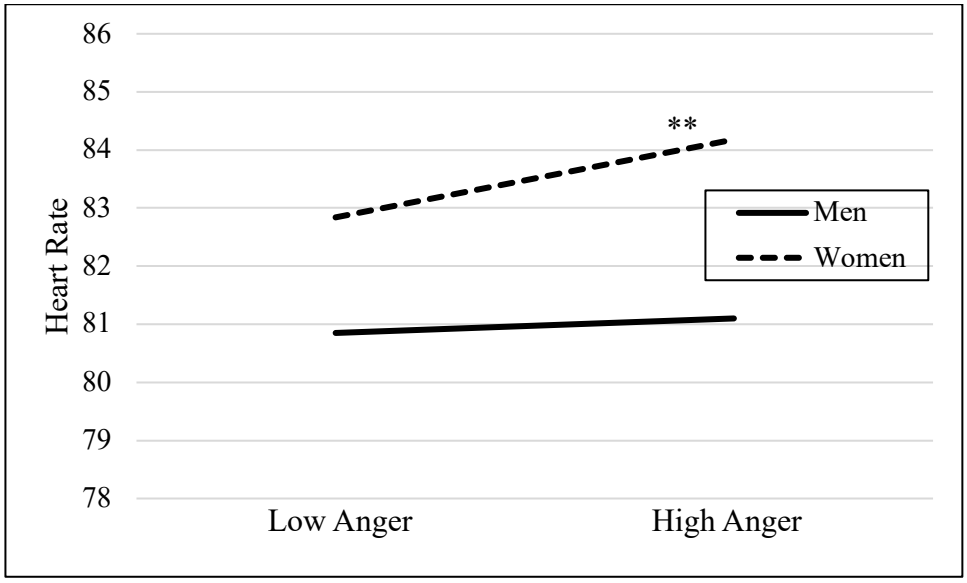

Figure 2. Interaction between momentary anger and sex predicting heart rate
Note. Simple main effects for anger at each sex are presented. **$p < .001$

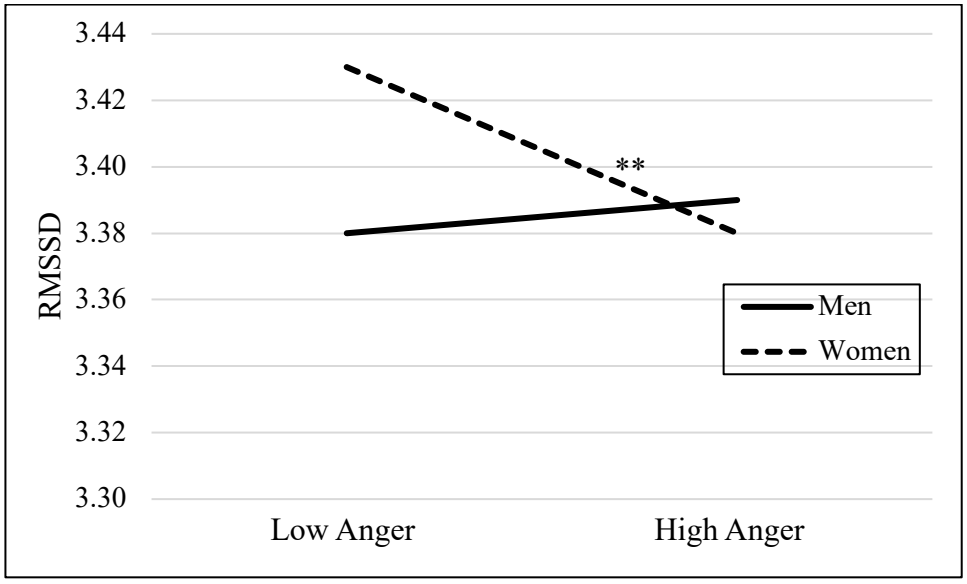

Figure 3. Interaction between momentary anger and sex predicting RMSSD
Note. Simple main effects for anger at each sex are presented. **$p < .001$

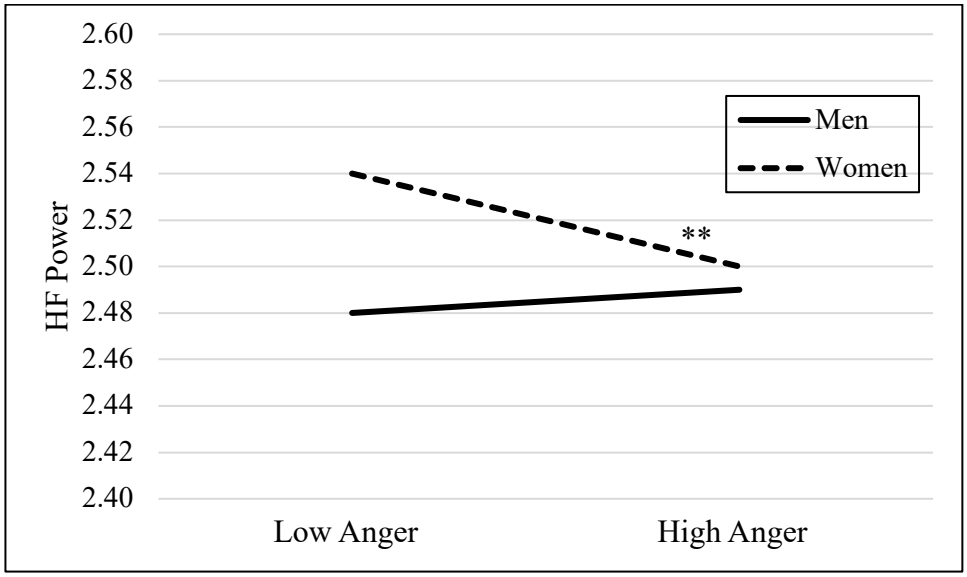

Figure 4. Interaction between momentary anger and sex predicting HF power
Note. Simple main effects for anger at each sex are presented. ***p* < .001

Figure 5. Interaction between momentary happiness and sex predicting heart rate
Note. Simple main effects for happiness at each sex are presented. ***p* < .001

Figure 6. Interaction between momentary happiness and sex predicting RMSSD
Note. Simple main effects for happiness at each sex are presented. **$p < .001$

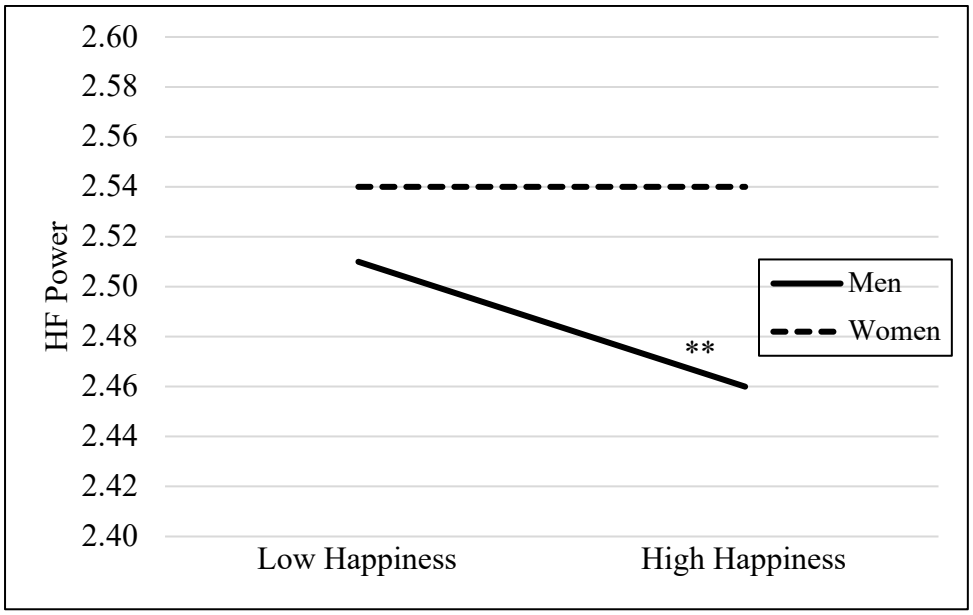

Figure 7. Interaction between momentary happiness and sex predicting HF power
Note. Simple main effects for happiness at each sex are presented. **$p < .001$

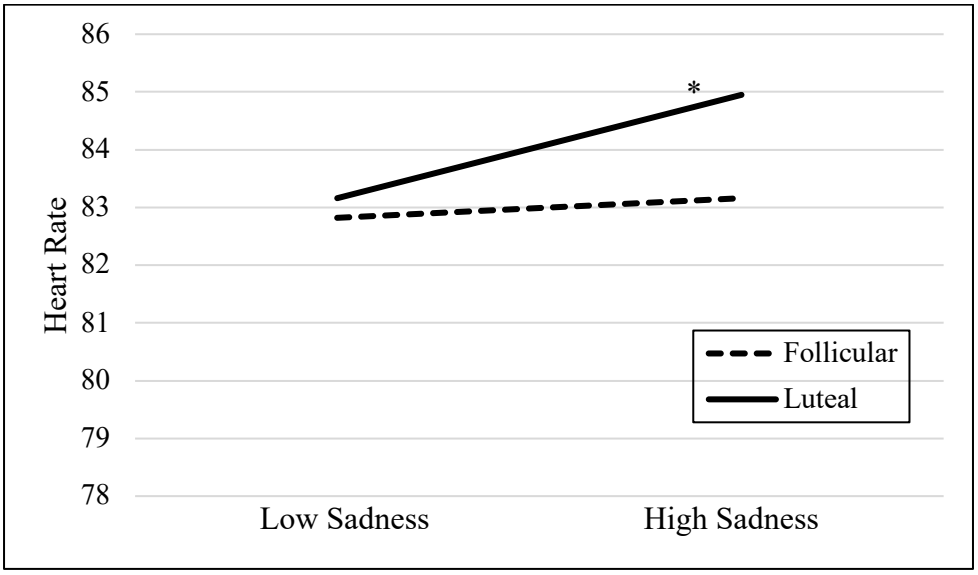

Figure 8. Interaction between momentary sadness and menstrual cycle phase predicting heart rate
Note. Simple main effects for sadness at each phase are presented. *p = .001

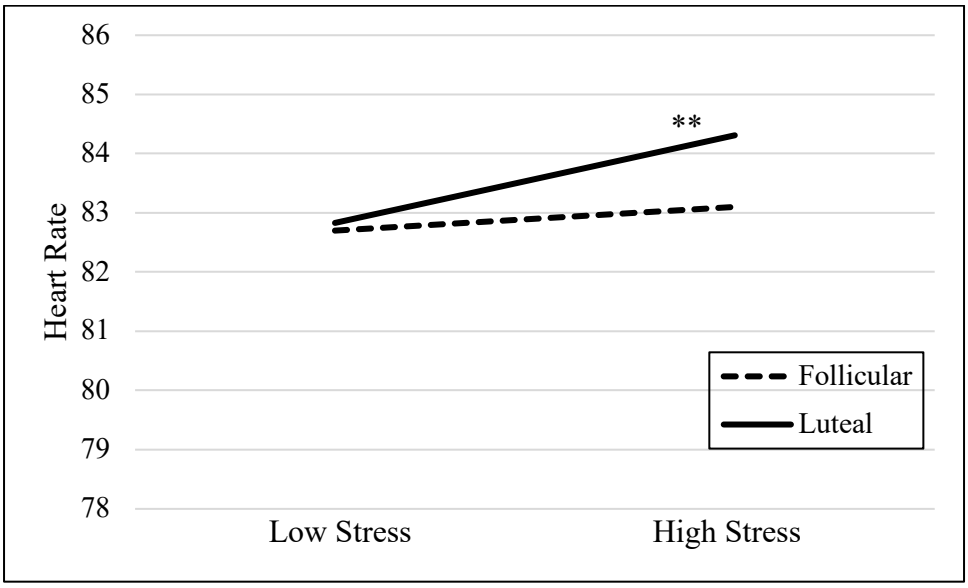

Figure 9. Interaction between momentary stress and menstrual cycle phase predicting heart rate
Note. Simple main effects for sadness at each phase are presented. **p < .001

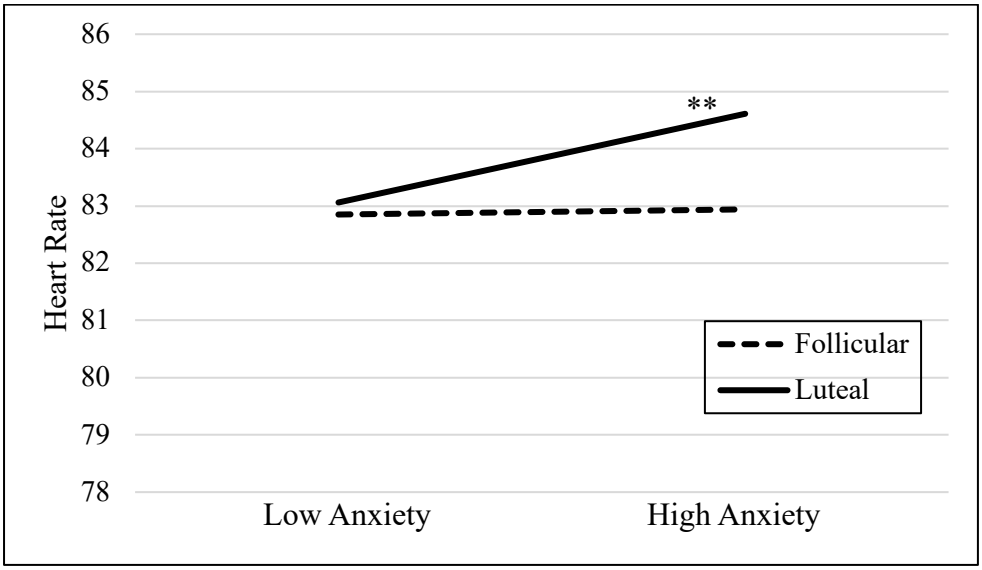

Figure 10. Interaction between momentary anxiety and menstrual cycle phase predicting heart rate
Note. Simple main effects for anxiety at each phase are presented. ***p* < .001

Figure 11. Interaction between momentary sadness and menstrual cycle phase predicting RMSSD
Note. Simple main effects for sadness at each phase are presented. **p* = .003

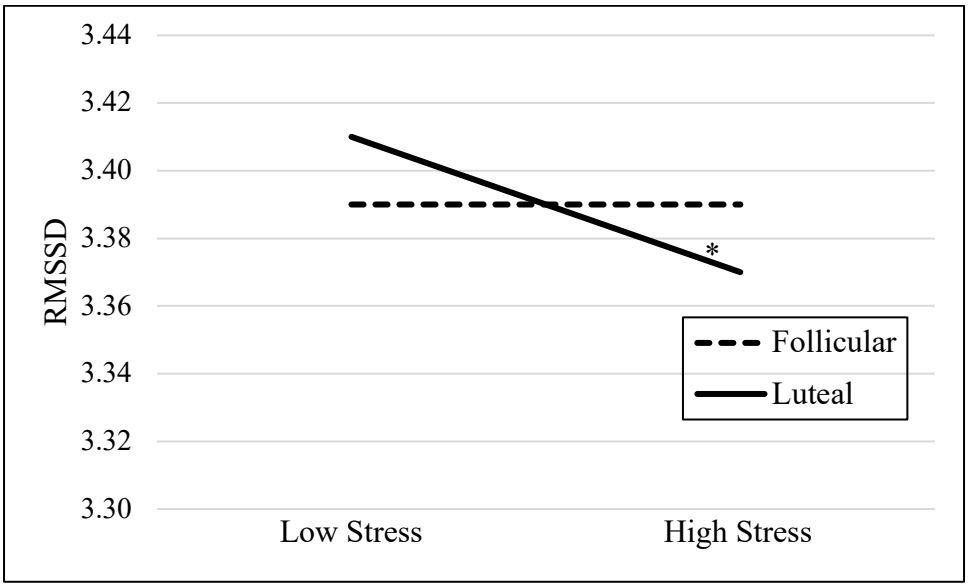

Figure 12. Interaction between momentary stress and menstrual cycle phase predicting RMSSD
Note. Simple main effects for stress at each phase are presented. *p = .004

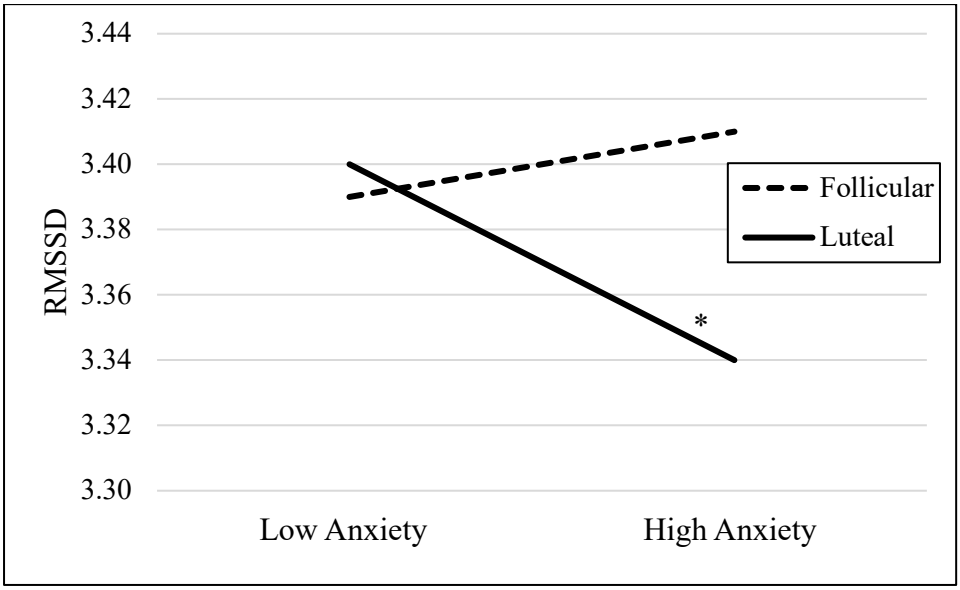

Figure 13. Interaction between momentary anxiety and menstrual cycle phase predicting RMSSD
Note. Simple main effects for anxiety at each phase are presented. *p = .002

Figure 14. Interaction between momentary sadness and menstrual cycle phase predicting HF power
Note. Simple main effects for sadness at each phase are presented. *p = .024

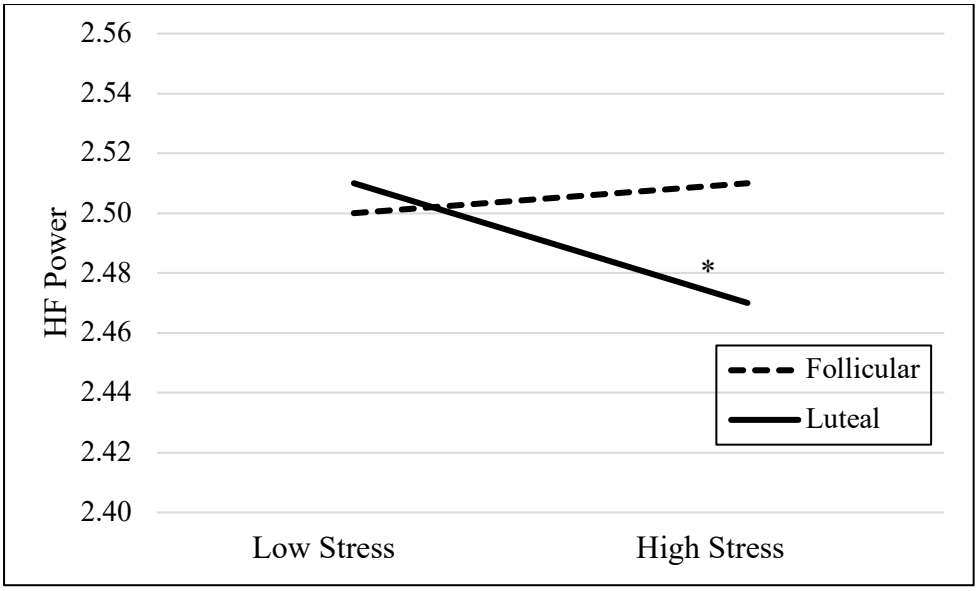

Figure 15. Interaction between momentary stress and menstrual cycle phase predicting HF power
Note. Simple main effects for stress at each phase are presented. *p = .001

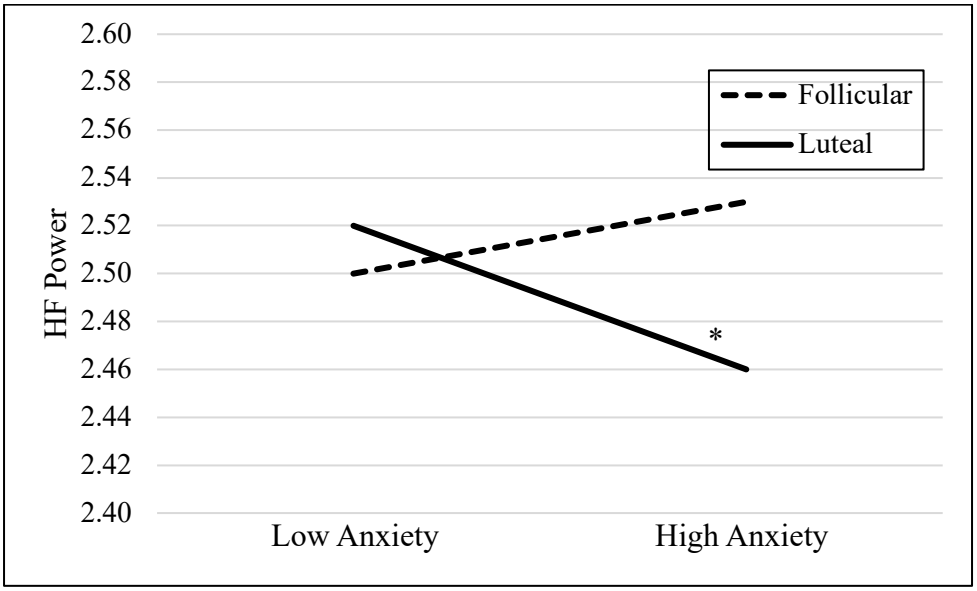

Figure 16. Interaction between momentary anxiety and menstrual cycle phase predicting HF power
Note. Simple main effects for anxiety at each phase are presented. *p = .004

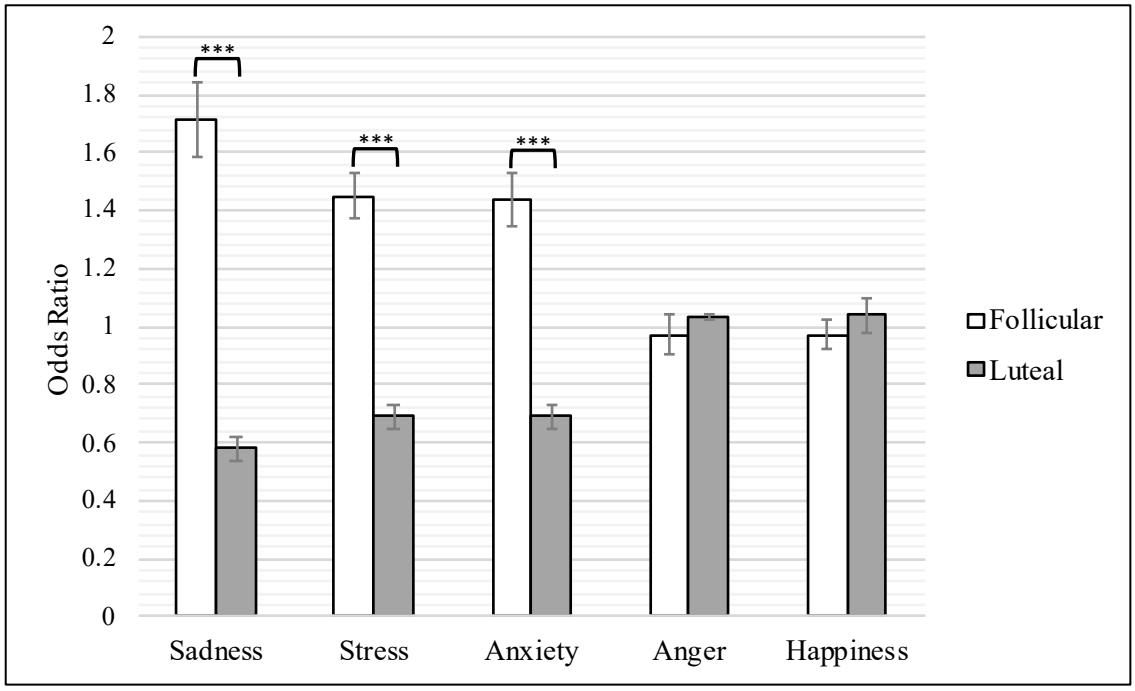

Figure 17. The odds of momentary emotions by menstrual cycle phase
Note. Odds ratios and corresponding standard errors are presented.